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Hypoxia Induced by Rapid Decompression from 8,000ft to 40,000ft - The Influence of Rate of Decompression

by

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ABSTRACT

Studies were made of the effects upon alveolar gases and electroencephalograms of rapid decompression from 8,000 feet at 1.6 and 12 sec with oxygen delivery 5 min before, immediately at, and 8 sec after the beginning of the decompression. The findings showed that hypoxia induced by delayed oxygen delivery was less on the slower decompression, although it was always severe, even when oxygen was delivered at the beginning of the decompression. A linear relationship was demonstrated between the intensity of the hypoxia, deduced from the changes of alveolar PO_2 and the increased activity (variance index) of the 8 - 16 Hz band of the frontal eeg.

Hypoxia; rapid decompression;

alveolar gas tensions; electroencephalogram.

INTRODUCTION

The occupants of modern high altitude aircraft are normally protected against the low pressure environment in which these aircraft fly by pressurisation of the cabin. Experience has shown, however, that either the structure of the pressure cabin or the pressure control system may fail during flight and thus the consequences of exposure to low environmental pressure must be considered. In certain operational and all modern transport aircraft the magnitude of the pressurisation is such that the cabin altitude does not exceed 8,000 ft when the aircraft is flying at its ceiling and in these circumstances it is usual for the occupants to breathe air throughout flight.

Several previous studies (Ernsting, Gedye and McHardy, 1960; Bryan and Leach, 1960) have demonstrated that if the altitude immediately after a sudden decompression exceeds 35,000 ft a significant impairment of performance will ensue, even if 100% oxygen is breathed immediately the decompression commences. In order to prevent this impairment of performance, the concentration of oxygen in the gas breathed before decompression must be raised by an amount which depends upon the pressure in the intact cabin (Ernsting, McHardy and Roxburgh, 1960; Ernsting 1963a). In the previous studies conducted in this laboratory the time of decompression employed was 1.5 to 2.0 sec. Although this duration of decompression can occur in certain operational aircraft, the shortest time of decompression which is likely in modern transport aircraft is of the order of 12 to 15 sec.

The present study was undertaken to determine the effect of increasing the duration of decompression over the range 8,000 ft to 40,000 ft from 2 sec to 12 sec upon the intensity of the consequent hypoxia. Oxygen was delivered to the subject's mask either immediately the decompression commenced or following a delay of 8 sec from the beginning of the decompression. Control experiments in which the subject breathed oxygen before, during and after the decompression

were also performed. The intensity of the hypoxia was determined by recording continuously the tensions of oxygen and carbon dioxide in the respired gas together with the electroencephalogram (eeg).

METHODS

Subjects

Three healthy male subjects, whose ages ranged from 20 to 38 years, were used.

Decompression Chamber

The subject was seated in the one man compartment of a three compartment decompression chamber. He was secured in the seat by means of an adjustable harness. The compartment could be rapidly evacuated through an orifice into a large reservoir by opening a pneumatically controlled valve. The pressure in the reservoir was reduced before each decompression to a value which allowed equilibration of the pressures in these two compartments of the chamber at the desired final value (141 mm Hg absolute). Orifices were used which provided durations of decompression (time for completion of 90% of the total pressure change) from a simulated altitude of 8,000 ft to one of 40,000 ft of 1.6 and 12.0 sec. A medical officer was held at an altitude of 8,000 ft in the lock to the subject's compartment.

Breathing Equipment

The subject wore an oronasal mask fitted with inlet and compensated outlet valves. A length of flexible hose 5 cm in diameter with a capacity of 2 litre was attached to the inlet port of the mask. When required, oxygen was passed into this hose at a point 2 cm upstream of the inlet port of the mask from a reducing valve placed outside the decompression chamber. The mass flow of oxygen, which was controlled by a metering orifice and a shut-off valve, was set

at 15 litre/min (at 760 mm Hg and 15°C). Preliminary experiment showed that this system delivered a volume flow of 80 L/min at an absolute pressure of 141 mm Hg.

Respiratory Measurements

The absolute pressure in the decompression chamber was recorded by means of a strain gauge transducer.

The expiratory volume was recorded by connecting the outlet port of the mask to a heated capillary flowmeter. The pressure difference created by flow through the flowmeter was measured with a strain gauge transducer. The amplified output of this transducer was integrated with respect to time and the integrated output recorded continuously. The output of the integrator was brought to 0 every 30 sec. The record of expiratory volume was calibrated before and after each experiment by passing known volumes of air through the flowmeter from a spirometer at ground level. Preliminary experiments had demonstrated that the output of the integrator for the passage of a given volume of gas through the flowmeter measured at the pressure of the environment was independent of the absolute pressure over the pressure range of 760 to 140 mm Hg.

The partial pressures of oxygen and carbon dioxide (P_{O_2} and P_{CO_2} respectively) in the gas flowing through the oronasal mask were recorded continuously by means of a respiratory mass spectrometer (Fowler and Hugh Jones, 1957). The instrument was mounted close to the external surface of the decompression chamber and the heated sampling line passed through the chamber wall. The tip of the sampling probe was placed in the cavity of the subject's oronasal mask.

The outputs of the instrument were calibrated by exposing the tip of the probe to gas mixtures containing known P_{O_2}s and P_{CO_2}s, both at a simulated altitude of 8,000 ft before each rapid decompression and immediately following the completion of each experiment at a simulated

altitude of 40,000 ft. The relationships between the P_{O_2} and P_{CO_2} in the dry gas entering the mass spectrometer and the deflections of the corresponding pen motor were found to be linear and, in the steady state, unaffected by reduction of environmental pressure. On rapid decompression from 8,000 ft to 40,000 ft in 1.6 sec, with the instrument sampling gas of a constant composition, the time taken for 90% of the response to the consequent fall of the partial pressure of a constituent was 3.2 sec.

Electroencephalogram

Three channels of eeg activity were recorded from four electrodes arranged antero-posteriorly over the subject's dominant cerebral hemisphere (electrode positions 5, 10, 15 and 18 or 3, 8, 13 and 17 in the 10/20 system). The electrodes consisted of fine hypodermic steel needles (26 swg) to each of which a fine wire had been soldered. An earth electrode was attached to one lower limb. The potential changes at the electrodes were amplified and recorded on a direct ink writer. The outputs were calibrated at the beginning and end of each experiment by applying a potential of 50 microvolts to the input of each amplifier channel. In order to maintain a constant level of alertness the subject performed a sequential motor task (Gedye, 1964) which involved the repetition of a previously learned sequence of 8 digital operations every 5 sec. The subject was able to monitor his own performance by a visual display.

Recording

The outputs of the various amplifiers were fed onto appropriate direct writing and bromide paper galvanometer recorders. In addition the electrical outputs representing pressure in the decompression chamber, integrated expiratory flow, P_{O_2}, P_{CO_2} and the three channels of eeg activity were fed onto magnetic tape. The magnetic recording tape was indexed before the experimental record was made (Goddard and Pennell, 1964). This tape index was presented visually to the

experimenters to allow subsequent identification of specific events. In order to ensure that the electrical outputs were being recorded satisfactorily on the magnetic tape the output from the play-back head of the tape recorder was connected through appropriate amplifiers to a 12-channel direct ink writer. The experimenters monitored this play-back record by means of a closed circuit television system.

Conduct of the Experiment

Each subject was exposed twice to each of six conditions. The six conditions were the two rates of rapid decompression (fast and slow), each of which was repeated thrice with varying times of oxygen delivery. Thus oxygen was delivered to the subject either 5 min before the rapid decompression (oxygen throughout), immediately the decompression occurred (oxygen early), or 8 sec after the commencement of the decompression (oxygen late).

The series of exposures of each subject were arranged in four sessions each consisting of three decompressions with a constant rate of decompression in a given session. The rates of decompression (slow and fast) were alternated for each consecutive session. In a given session of exposures oxygen was delivered at each of the specified times once. The order of oxygen delivery times was randomised using a balanced design.

Before each exposure to reduced pressure the acceptability of the quality of the recording of the outputs of the flow integrator, the respiratory mass spectrometer and the eeg were checked at ground level. The subject was then decompressed in 30-60 sec to a simulated altitude of 8,000 ft. A period of 5 min was spent at this altitude with the subject breathing either air or oxygen. During the initial part of this period the subject was presented with a new pattern to learn on the motor task. This he repeated until he considered that he was proficient at the task. At the end of this period the various

recorders, including the magnetic tape recorder, were set in motion and the acceptability of the recordings checked. The subject was instructed to close his eyes for a period of about 10 sec and the eeg record was inspected for the appearance of alpha activity. When the subject opened his eyes the motor task was started and he completed a series of at least 12 complete operations, each of which lasted 5 sec.

At the completion of the 60 sec control period at the motor task, the subject was decompressed to a simulated altitude of 40,000 ft. He was warned of the instant at which the decompression would commence by a 5 point count-down. The subject was instructed to breathe out during the latter part of the count-down so that when the decompression occurred his glottis was open and the volume of gas within the respiratory tract was relatively small. The motor task was restarted automatically at the beginning of the decompression. If the subject had been breathing air before the decompression the oxygen supply to his mask was turned on at the appropriate time. The subject continued performing the task for 90 sec after the rapid decompression. The recordings were completed by instructing the subject to shut his eyes for approximately 10 sec and calibrating the outputs of the respiratory mass spectrometer. The decompression chamber was then recompressed to ground level at a rate of between 15,000 and 20,000 ft/min and the symptoms, if any, experienced by the subject, recorded. The subject breathed air during the latter part of the descent to ground level. In each session of 3 decompressions a period of at least 15 min separated the end of one exposure to reduced pressure from the beginning of the next. Each session of three decompressions was separated from the preceding session by at least 18 hours.

RESULTS

Symptoms

The three subjects successfully completed the series of rapid decompressions comprising the investigation. The symptoms, reported

by the subjects consisted of varying degrees of disturbance of consciousness and the occasional occurrence of abdominal discomfort or pain. The central nervous symptoms usually appeared approximately 15 sec after the beginning of the rapid decompression and disappeared before the end of the first minute at 40,000 ft. The commonest symptom was mild confusion which was variously described as light-headedness or dizziness. On occasions there was partial loss of vision, usually in the periphery of the visual fields. The intensities of the confusion and visual disturbances were assessed as either mild (+) or severe (++). The intensity and nature of the symptoms varied with the experimental condition. The incidence of symptoms is summarised in Table 1.

TABLE 1

Incidence and Nature of Central Nervous Symptoms Occurring after Rapid Decompression

Condition	No of decompressions with symptoms	No of decompressions with specific symptoms			
		Confusion		Impairment of vision	
		+	++	+	++
Fast decompression					
Oxygen throughout	0	-	-	-	-
Oxygen early	5	5	-	3	-
Oxygen late	6	1	5	2	2
Slow decompression					
Oxygen throughout	0	-	-	-	-
Oxygen early	5	5	-	2	-
Oxygen late	5	2	3	2	-

(There was a total of 6 decompressions to each experimental condition)

Abdominal symptoms occurred in 9 out of the 36 experiments. These symptoms arose during or immediately after the rapid decompression to 40,000 ft and usually remained throughout the duration of the exposure to this altitude. They were relieved by recompression. The intensity of the symptoms varied from one subject to another and with the rate of decompression. There was no correlation between occurrence of abdominal symptoms and the time at which the first breath of oxygen was delivered. No case of severe incapacitating abdominal pain occurred in this study and the intensity of this symptom was classified as either mild or moderate (Table 2).

TABLE 2

Incidence of Abdominal Symptoms after Rapid Decompression

Subject	No of decompressions with abdominal symptoms			
	Fast decompression		Slow decompression	
	Mild	Moderate	Mild	Moderate
DD	0	0	0	2
FP	2	0	4	1
DIF	0	0	0	0

Alveolar Ventilation

After the individual values of the tidal volume had been corrected to the pressure and temperature conditions existing within the respiratory tract (btps), the values of the pulmonary ventilation were calculated for various periods in each experiment. The number of breaths in each period was also determined. The corresponding alveolar ventilation was calculated using an assumed value for the total respiratory dead space. It was assumed that the subject's respiratory dead space expressed in ml was equal to his weight expressed in lb (Donevan, Palmer, Varvis and Bates, 1959) and that the effective dead space of the oronasal mask was 80 ml. Alveolar ventilation was calculated in this manner for the 60 sec period during which the

subject performed the motor task at 8,000 ft, for the 30 sec period immediately before the beginning of the rapid decompression, for the period 16 - 45 sec after the beginning of decompression and for the period 61 - 90 sec after the beginning of the decompression. The mean values of the alveolar ventilation for the exposures of the subjects to each of the six experimental conditions are presented in Table 3. In all the conditions the alveolar ventilation was greater during the period immediately before the rapid decompression than during the period in which the control task was carried out. The alveolar ventilation increased immediately after the rapid decompression. This increase was considerably greater when oxygen was not breathed until after the beginning of the rapid decompression than when oxygen was breathed for 5 min before the decompression. The values of alveolar ventilation obtained at 40,000 ft during the third half-minute period were similar in all the experimental conditions and were the lowest recorded throughout the exposures.

TABLE 3

Alveolar Ventilation before and after Rapid Decompression

Condition	Alveolar ventilation l(btps)/min			
	Before rapid decompression		After rapid decompression	
	Control task	-30 to 0 sec*	16 to 45 sec*	61 to 90 sec*
Fast decompression				
Oxygen throughout	9.0	10.9	10.4	7.0
Oxygen early	7.5	9.6	12.7	5.8
Oxygen late	7.1	7.8	11.6	6.0
Slow decompression				
Oxygen throughout	7.1	9.0	10.2	6.0
Oxygen early	6.4	8.3	12.2	5.6
Oxygen late	6.3	8.3	12.7	5.6

* Time measured from commencement of rapid decompression

Respired Gas Tensions

The rapid decompression produced a rapid fall of both P_{O_2} and P_{CO_2}. The P_{O_2} remained low with only a small difference between the inspired and expired values until the inspiration following the instant at which the oxygen supply was turned on. The P_{O_2} in the expired gas then increased rapidly to attain a relatively steady value. The P_{CO_2} also increased following the rapid decompression to a relatively steady value. With the rapid rate of decompression gas flowed from the respiratory tract throughout the decompression. In the slow decompressions, however, inspiration frequently occurred whilst the pressure in the chamber was still falling. Although the oxygen supply was turned on either immediately the decompression began or 8 sec after, oxygen could not enter the respiratory tract until the following inspiration occurred. The interval between the beginning of the decompression and the first inspiration of oxygen was measured for each experiment and the mean values for each condition are presented in Table 4.

TABLE 4

Time of First Inspiration of Oxygen

Condition	Mean interval between beginning or rapid decompression and first inspiration of oxygen (sec)
Fast decompression	
Oxygen early	5.5 (2.0)
Oxygen late	10.8 (1.1)
Slow decompression	
Oxygen early	6.1 (2.9)
Oxygen late	11.0 (1.3)

Values in parentheses are ± standard deviation

The composition of the gas in the mask cavity at the end of expiration was assumed to represent that of the alveolar gas. Owing, however, to the relatively long response time of the mass spectrometer to changes in the tension of water vapour, and to the temperature in the mask being lower than that of the alveolar gas, it was necessary to apply a correction factor to the measured gas tensions to obtain the corresponding values in the alveolar gas. The magnitude of the correction was calculated from the observed end tidal P_{O_2} and P_{CO_2} obtained at 40,000 ft in the experiments in which oxygen was breathed for 5 min before decompression. In these experiments the tension of nitrogen in the expired gas amounted to only 2 - 4 mm Hg and it was assumed that the difference between the sum of the observed P_{O_2} and P_{CO_2} and the total absolute pressure in the mask (141 mm Hg) was due to water vapour. The mean value of this difference was determined for all the breaths during the exposures to 40,000 ft in which oxygen had been breathed for 5 min before decompression. This mean value was 31.3 mm Hg. The factor by which the measured P_{O_2} and P_{CO_2} were multiplied to give the corresponding values in the alveolar gas was obtained by the calculation

$$\text{Correction factor} = \frac{P_B - P_{A_{H_2O}}}{P_B - P_{M_{H_2O}}}$$

where P_B = = environmental pressure

$P_{A_{E_2O}}$ = water vapour pressure at body temperature

$P_{M_{H_2O}}$ = recorded water vapour tension in expired gas

The correction factor calculated in this manner was 0.972 at 8,000 ft and 0.855 at 40,000 ft.

The corrected values of the alveolar oxygen and carbon dioxide tensions ($P_{A_{O_2}}$ and $P_{A_{CO_2}}$ respectively) for each breath were plotted against time for each individual experiment and a smooth curve was fitted to each individual set of data by eye. The values of $P_{A_{O_2}}$ and $P_{A_{CO_2}}$ were then read from the curves at intervals of 5 sec from 25 sec

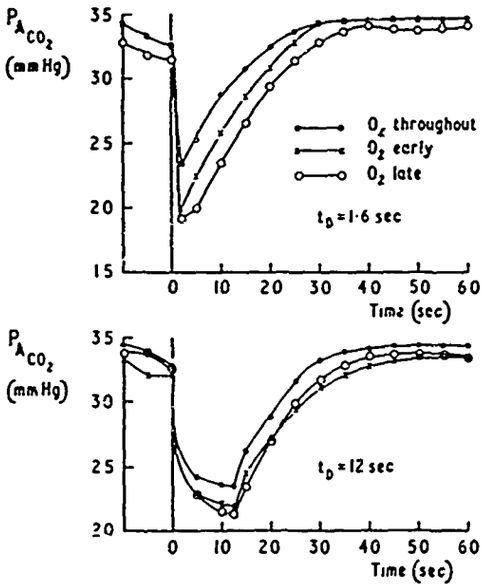

Fig. 1. Effect of rapid decompression from 8,000 feet to 40,000 feet in 1.6 sec (upper curves) and 12 sec (lower curves) upon alveolar carbon dioxide tension under three conditions of oxygen delivery viz: (a) oxygen breathed for 5 min before the rapid decompression and throughout the subsequent period at 40,000 feet (closed circles); (b) air breathed before the decompression and oxygen delivered immediately the rapid decompression began (crosses); (c) air breathed before the decompression and oxygen delivered 8 sec after the beginning of the rapid decompression (open circles). The rapid decompression commenced at time 0 and each point represents the mean of 6 experiments.

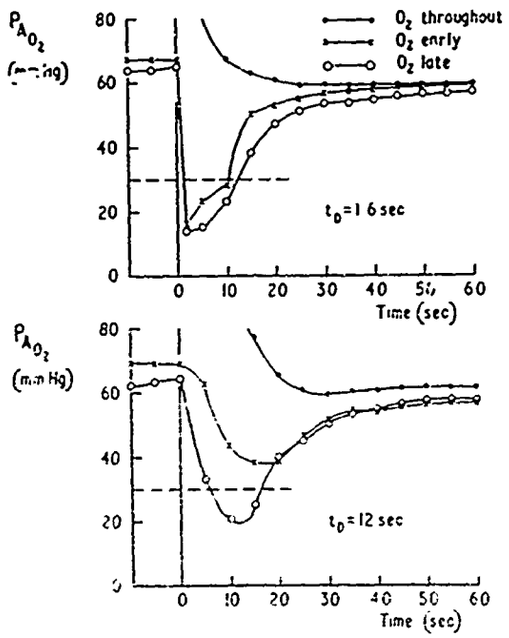

Fig. . Effect of rapid decompression from 8,000 feet to 40,000 feet in 1.6 sec (upper curve) and 12 sec (lower curves) upon alveolar oxygen tension under three conditions of oxygen delivery viz: (a) oxygen breathed for 5 min before the rapid decompression and throughout the subsequent period at 40,000 feet (closed circles); (b) air breathed before the decompression and oxygen delivered immediately the rapid decompression began (crosses); (c) air breathed before the decompression and oxygen delivered 8 sec after the beginning of the rapid decompression (open circles). The rapid decompression commenced at time 0 and each point represents the mean of 6 experiments.

before to 90 sec after the beginning of the rapid decompression. The data obtained in this manner were then grouped together according to the experimental conditions and analyses of variance performed. The means of the six values obtained for each instant in time in a given experimental condition were also calculated. The curves representing these mean values are presented in Figs 1 and 2.

a. Alveolar Carbon Dioxide Tension

The P_{ACO_2} fell progressively with time during the period at 8,000 ft before the rapid decompression, reaching a mean value of 32.4 mm Hg at the beginning of the decompression. There was no significant difference between the mean values of P_{ACO_2} obtained for air and oxygen breathing at 8,000 ft. The minimum value of the P_{ACO_2} (Table 5) occurred on the first breath after the decompression in the short duration decompressions but it was delayed by about 10 sec in the slow decompressions.

Although the P_{ACO_2} reached a lower minimum value in the fast decompressions it remained lower for a longer time in the slow series than in the fast series (Fig 1). Beyond 40 sec after the decompression there was no significant difference between the values of P_{ACO_2} in the various experimental situations. With both rates of decompression the time of delivery of oxygen affected the pattern of P_{ACO_2} change (Fig 1). The lowest values of P_{ACO_2} occurred when oxygen was delivered after decompression and the highest when oxygen was breathed throughout the experiment (up to 20 sec after decompression the differences were significant at $P = 0.01$).

b. Alveolar Oxygen Tension

The P_{AO_2} was not recorded before the rapid decompression when oxygen was breathed throughout the experiment. On decompression the P_{AO_2} fell gradually to attain a new steady value some 25-30 sec after the beginning of the decompression (Fig 2). This value did not differ significantly from the mean for all experiments over the last 30 sec at 40,000 ft (58.5 mm Hg).

When air was breathed at 8,000 ft the $P_{A_{O_2}}$ increased progressively with time to reach a mean value of 66.7 mm Hg immediately before the decompression. On decompression the $P_{A_{O_2}}$ fell rapidly. In the fast decompressions the minimum $P_{A_{O_2}}$ was reached immediately after the decompression (Fig 2). The recovery of $P_{A_{O_2}}$ was slower when oxygen delivery was delayed than when it occurred at the instant of decompression (the mean $P_{A_{O_2}}$ differences were significant up to 20 sec after decompression, P = 0.05). When oxygen was delivered during the slow decompressions the $P_{A_{O_2}}$ fell to a minimum value at the end of the decompression. The values of $P_{A_{O_2}}$ when oxygen delivery was delayed were significantly lower than those produced by the immediate delivery of oxygen for up to 15 sec after decompression (P = 0.001).

TABLE 5

The Minimum Alveolar Carbon Dioxide and Oxygen Tensions following Rapid Decompression

Condition	Mean minimum P_{ACO_2} (mm Hg)	Mean minimum $P_{A_{O_2}}$ (mm Hg)
Fast decompression		
Oxygen throughout	23.3	57.5
Oxygen early	19.7	15.8
Oxygen late	19.3	14.0
Slow decompression		
Oxygen throughout	22.7	58.3
Oxygen early	21.3	27.5
Oxygen late	21.0	18.7

Electroencephalogram

The eeg recorded on magnetic tape was subsequently analysed using an analogue computer. The electrical signal recorded from a pair of electrodes was analysed by passing it through each of a series of

filter sections (with terminal slopes of 30 db/octave) which gave frequency bands of 1-2, 2-4, 4-8, 8-16 and 16-32 Hz. The outputs from the filter sections were squared to render them unidirectional and then continuously integrated. The integrated output for each of the frequency bands was recorded simultaneously against time. These computations gave the signal variance, the slope of each integrated output being proportional to the eeg activity in that band. Changes in the slope of a variance curve indicated a change of eeg activity in that frequency band and channel.

A typical record of the analysis of the anterior channel of the eeg from an experiment in which the decompression was fast and oxygen was delivered at the beginning of the decompression is presented in Fig 3. The pressure in the decompression chamber and the P_{O_2} in the mask cavity are also shown. There was a marked increase in the slope of the variance signal for the 8-16 Hz band when the subject shut his eyes at the beginning and at the end of the experiment. During the period in which the task was performed at 8,000 ft, the slopes of the integrals were relatively constant. The slopes remained unchanged during and immediately after the rapid decompression. Some 15 sec after the beginning of the decompression, however, the slope of the variance signal in the 8-16 Hz band suddenly became steeper. After a further 10 sec the slope decreased progressively until the original slope was attained.

The change of the activity in a given frequency band was expressed as a variance index (Byford, 1965). This index was calculated by dividing the value of the increased slope which occurred after the decompression by the slope obtained in the same frequency band during the control period at 8,000 ft before the decompression occurred. In the majority of the experiments in which an increase of eeg activity was seen after decompression, the increase occurred in the 8-16 Hz band alone. The mean values obtained for the variance index in the 8-16 Hz band for each of the conditions studied are presented in Table 6. When oxygen was breathed throughout an experiment there was virtually no change of activity in the 8-16 Hz

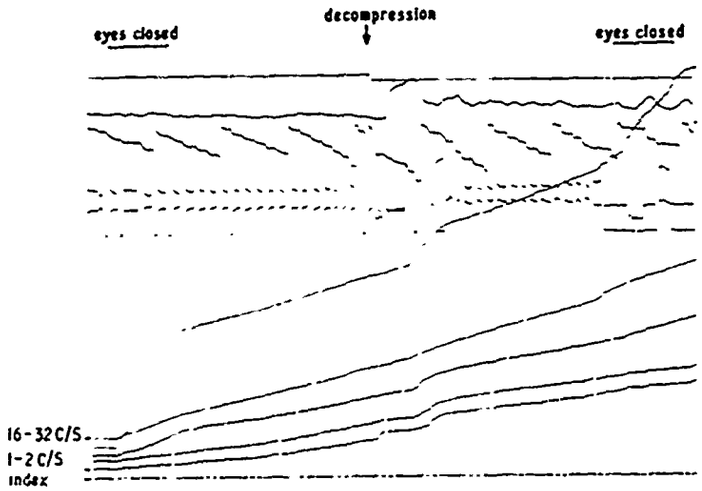

Fig 3. Effect of rapid decompression from 8,000 feet to 40,000 feet in 1.6 sec with oxygen delivered at the beginning of the rapid decompression upon the anterior channel of the eeg. Traces reading from above downwards are (a) pressure in the decompression chamber (P_B); (b) output of ear oximeter S_{aO_2} (not analysed in this paper); (c) integrated expired gas flow ($\dot{V}E$); (d) PO_2 in mask cavity; (e) PCO_2 in mask cavity; (f) to (j) variance of eeg signal for the frequency bands 16 - 32 (f), 8 - 16 Hz (g), 4 - 8 Hz (h), 2 - 4 Hz (i) and 1 - 2 Hz (j); (k) tape index. Note the rapid increase in signal variance in the 8 - 16 Hz band when the eyes were closed at the beginning and the end of the experiment. The variance in this band also increased in the period 15 to 25 sec after the rapid decompression.

band during or after decompression. When air was breathed at 8,000 ft before decompression there was frequently an increase in activity in all three channels after the decompression. The increase was usually least in the posterior channel. At either rate of decompression the variance index was on average lower when the oxygen was delivered at the instant of decompression than when it was delivered after a delay of 8 sec.

TABLE 6

Mean Values for Variance Index $\left(\frac{\text{eeg activity after decompression}}{\text{eeg activity in control period}}\right)$ for Frequency Band 8-16 Hz

Condition	Mean variance index for 8-16 Hz		
	Anterior Channel	Middle Channel	Posterior Channel
Rapid decompression			
Oxygen throughout	1.0	1.0	1.2
Oxygen early	2.2	2.6	2.1
Oxygen late	4.5	5.2	2.9
Slow decompression			
Oxygen throughout	1.0	1.1	1.0
Oxygen early	1.4	2.1	1.9
Oxygen late	2.6	2.2	2.0

The delay between the beginning of the decompression and the first appearance of an increase in the signal variance in the 8-16 Hz band was measured for each exposure. This interval was found to be unaffected by the rate of decompression or the time at which oxygen was delivered. The mean interval between the beginning of the decompression and the beginning of the increase of eeg activity was 15.2 sec (with a SD of $\pm$0.8 sec).

When oxygen delivery was delayed for 8 sec after a fast decompression there was also an increase of eeg activity in the lower frequency bands. The analysis of a typical experiment is presented in

Fig 4. Some 15 sec following the decompression there was a sudden increase of activity in the 1-2, 2-4 and 4-8 Hz bands as well as in the 8-16 Hz band. The large increase in activity in the 1-2 and 2-4 Hz bands ceased almost as rapidly as it had begun some 15 sec later.

DISCUSSION

Alveolar Ventilation and Respired Gas Tensions

The alveolar ventilation recorded during the control period before rapid decompression at 8,000 ft (Table 3) was raised, as compared with the value normally obtained in resting seated subjects (5.0 - 6.0 l(btps)/min). This alveolar hyperventilation was reflected in the low resting values obtained for the $P_{A_{CO_2}}$ during this period (Fig 1). These changes, which were accentuated during the 30 sec immediately preceding the decompression, were independent of the gas breathed during this period. They were due to the subject's awareness that decompression was imminent. This hyperventilation increased the $P_{A_{O_2}}$ slightly above that normally found in resting subjects exposed to breathing air at 8,000 ft, so that the severity of the hypoxia induced by the subsequent rapid decompression may have been slightly less than would have been the case in the absence of the hyperventilation.

Although, when air was breathed until the decompression to 40,000 ft, the hose attached to the inlet port of the mask was filled with oxygen at a fixed time in relation to the beginning of decompression, the actual time at which oxygen first entered the respiratory tract varied from one experiment to another. Oxygen did not enter the respiratory tract until the inspiration following the point at which the oxygen supply was turned on. In none of the fast decompressions did the subject inspire until the decompression was complete, but with the slow rate of decompression most subjects had taken at least one inspiration before the fall in environmental pressure had ceased (Table 4). It is difficult to assess how the instructions given to each subject to breathe out immediately before and during the

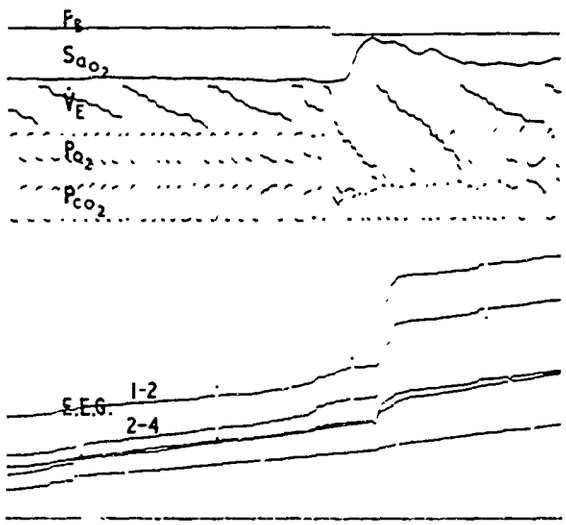

Fig 4. Effect of rapid decompression from 8,000 feet to 40,000 feet in 1.6 sec with oxygen delivery delayed to 8 sec after the beginning of the decompression upon the anterior channel of the eeg. The order of presentation of the traces in this figure is the same as for Fig 3. Note the rapid increase in the signal variance in the 1 - 2 Hz and 2 - 4 Hz bands of the eeg in the period 15 - 30 sec after the rapid decompression.

decompression influenced his breathing pattern during and after the decompression. It is unlikely that the instructions had any influence in the fast series of decompressions, but they may have contributed to the delay between the beginning of decompression and the first inspiration in the slow series. In the breathing equipment used in this study the dead space between the point of entry of the oxygen supply and the nose and mouth was small, amounting to only 100 ml. Thus the composition of the gas entering the respiratory tract on the first inspiration after oxygen had been turned on approached 100% oxygen, and all subsequent breaths were 100% oxygen.

The decompression to 40,000 ft produced a marked reduction of the P_{ACO_2} and P_{AO_2} (Figs 1 and 2). The primary cause of the reduction of these gas tensions was the fall of the total gas pressure within the respiratory tract. The pattern of change of P_{ACO_2} in this situation is determined by the relationships between the change of total alveolar gas pressure, the alveolar ventilation and the rate at which carbon dioxide enters the alveolar gas from the pulmonary capillary blood (and the lung parenchyma). This rate of excretion of carbon dioxide depends in turn upon the P_{CO_2} in the mixed venous blood and the cardiac output.

The rate of fall of P_{ACO_2} induced by decompression was related directly to the rate of fall of environmental pressure, although this did not affect the minimum value reached (Table 5). The influence of the time of delivery of oxygen upon the time course and magnitude of the changes of P_{ACO_2} induced by a given rate of decompression was due to the increased degree of alveolar hyperventilation associated with delay in the delivery of oxygen. When the hypoxic stimulus to respiration had been removed by breathing oxygen at 40,000 ft, alveolar ventilation fell to the minimum recorded during the experiment (Table 3), and this change was associated with a rise of the P_{ACO_2} (Fig 1).

When oxygen was breathed at 8,000 ft before decompression the P_{AO_2} did not fall below 55 mm Hg after the decompression (Table 5).

The prolongation of the fall of P_{AO_2} following the completion of the decompression in this experimental condition was due to the concomitant rise of P_{ACO_2} (Fig 2). There was a considerable difference between the minimum values of the P_{AO_2} induced by the two rates of decompression when oxygen was delivered at the beginning of the decompression (Table 5). Whilst no subject inspired during the rapid decompressions, in the slow decompression each subject took at least one breath of oxygen whilst the environmental pressure was still falling. Thus, in the slow decompressions, replacement of nitrogen by oxygen had begun before the minimum environmental pressure had been reached. The difference between the mean values of the minimum P_{AO_2} produced by the two rates of decompression was less marked when the delivery of oxygen was delayed until 8 sec after decompression. In this situation with the slow rate of decompression most of the fall of environmental pressure was complete before the first breath of oxygen was taken, so that the intrapulmonary pressure just before oxygen was breathed was similar with the two rates of decompression.

The intensity of the hypoxia induced by rapid decompression to 40,000 ft when air was breathed at 8,000 ft may be expressed in terms of the associated changes of P_{AO_2}. Since both the magnitude and the time course of the reduction of P_{AO_2} varied with the experimental condition, and even within the individual exposures to the same condition, the expression for the intensity for the hypoxia must take account of both the degree and the duration of the reduction of the P_{AO_2}. A useful measure of the intensity of hypoxia in this context is the time integral of the difference between the observed P_{AO_2} and a stated value of P_{AO_2} over the period for which the former is less than the latter. Considerations which are presented later in this paper led to the selection of the area on the P_{AO_2} - time plot bounded below by the observed curve and above by a horizontal line representing a P_{AO_2} of 30 mm Hg. The value of this area was determined from the P_{AO_2} - time plot for each experiment and the individual results are presented in Table 7. There was a considerable variation in the

intensity of the hypoxia produced by a given experimental condition. It is apparent, however, that of the four conditions in which air was breathed before decompression the hypoxia was least when oxygen was delivered at the beginning of a slow decompression and greatest when it was not delivered until 8 sec after the beginning of a fast decompression. Using this measure, the intensity of the hypoxia induced by oxygen delivery at 8 sec in a slow decompression was comparable with that associated with the delivery of oxygen at the beginning of a fast decompression and lay between the extremes found in the two other conditions.

TABLE 7

Intensity of Hypoxia - Values of Area below P_{AO_2} = 30 mm Hg on P_{AO_2} - Time Curve

Subject	Area below P_{AO_2} = 30 mm Hg on P_{AO_2} - time curve (mm Hg sec)			
	Fast decompression		Slow decompression	
	Oxygen early	Oxygen late	Oxygen early	Oxygen late
FF	60	140	6	72
FP	20	106	92	54
DF	58	126	14	28
DF	26	180	0	58
DD	74	100	8	30
LL	54	58	0	136
Mean	48	136	20	64

Subjective Disturbances

The absence of any subjective central nervous system disturbance when oxygen was breathed throughout an experiment (Table 1) showed that the fall of P_{ACO_2} produced by rapid decompression was not the primary cause of the impairment of consciousness and vision which occurred when air was breathed before the decompression. With a given rate of decompression, the increase of the intensity of hypoxia associated with delaying the delivery of oxygen from the beginning of the decompression to 8 sec later was associated with a greater degree

of confusion. Even in the experimental condition which gave the least degree of hypoxia as measured from the changes of P_{AO_2}, ie a slow decompression with the immediate delivery of oxygen, confusion and the dimming of vision were reported. Although dimming of vision occurred in a proportion of each of the conditions in which air was breathed before decompression, gross impairment of vision only occurred when oxygen delivery was delayed until 8 sec after the beginning of a rapid decompression.

Electroencephalographic Changes

Earlier studies (Ernsting, 1963b) of the effects of the hypoxia produced by rapid decompression to 40,000 ft whilst breathing air showed that the first eeg change consisted of the appearance of activity with a frequency of 8 to 13 Hz. Inspection of the records obtained in these experiments suggested that the amount of eeg activity was related to the intensity of the hypoxia as judged from the changes of the P_{AO_2}. The advantages of the form of analysis of the eeg employed in the present study (Byford, 1965) are that it gives a quantitative expression for changes of eeg activity and it is possible to time accurately changes of activity and relate them to other physiological events.

The changes of eeg activity which were recorded in the present investigation occurred in the 8-16 Hz band principally in the anterior and middle channels (Table 6). Some increase in activity in this band was also seen in the posterior channel, particularly with the more intense hypoxia. Care was taken to ensure that there was a constant level of visual tension throughout each experiment. Attention was only grossly reduced when, in some of the fast decompressions with oxygen delivery at 8 sec, vision was lost completely.

The eeg changes associated with the production of a uniform visual field occur principally over the posterior part of the head. Furthermore, the absence of any change of eeg activity in the control

experiments, in which oxygen was breathed throughout to prevent any increase of hypoxia, gives support to the hypothesis that the increase of eeg activity in the 8-16 Hz band seen in these experiments was due to cerebral hypoxia. The results of the control experiments also illustrate that as in the case of subjective disturbances the observed increase of eeg activity was not primarily due to hypocapnia.

The eeg showed no increase of activity until 15 sec after the beginning of decompression. The magnitude of this delay was unaffected by the rate of decompression and the time at which oxygen was delivered. The delay consisted of the transit time for blood with a low oxygen content to pass from the pulmonary capillaries to the capillary bed in the relevant region of the brain, the time taken for cerebral metabolism to reduce the cerebral tissue P_{O_2} to the level at which an increase of eeg activity occurs and the latency of the eeg response to the low P_{O_2}. Measurements of the transit time from the pulmonary capillaries to the ear lobe, made by recording the oxygen saturation of the blood flowing through the ear following a rapid decompression whilst breathing air, gave a mean transit time 6.1 sec. The transit time to the cerebral capillary bed may be slightly less than this value, but this factor accounts for at least a third of the total delay from the beginning of decompression to the first change of eeg activity. The oxygen store of the brain has been estimated by Kety (1950) to amount to approximately 7 ml. At the normal rate of oxygen consumption this store would be exhausted in about 8 sec and thus the cerebral tissue oxygen tension would be expected to fall to a very low value within 6 - 8 sec of the arrival of deoxygenated blood in the cerebral capillaries. There is no information available with regard to the latency of the eeg response to a reduction of cerebral tissue oxygen tension. It is not possible therefore to distinguish the relative contributions of the depletion of the cerebral oxygen store and the latency of the eeg response to the total 15 sec delay.

It is apparent from inspection of Table 6 that the magnitude of the increase of activity in the 8-16 Hz band of the eeg following

rapid decompression, as indicated by the mean values of the variance index, was related to the intensity of the hypoxia induced by the specific experimental conditions. Thus, the conditions giving rise to the greatest degree of hypoxia as judged both by the magnitude of the fall of the P_{AO_2} (Fig 2) and by the symptoms (Table 1), ie the late delivery of oxygen after the fast decompression, produced on average the largest increase of the variance index. Various estimates of the degree of hypoxia have been explored in an attempt to correlate the changes of the variance index with the hypoxia. Only a poor correlation was obtained when either the minimum P_{AO_2} or the value of the P_{AO_2} at any of several time intervals following the initiation of the rapid decompression was used as a measure of the intensity of the hypoxia.

Since, as pointed out earlier in this discussion, the measure of the intensity of the hypoxia must take account not only of the degree but also the duration of the reduction of P_{AO_2} the value of using the area on the P_{AO_2} - time plot bounded by the observed curve and a horizontal line representing a specified P_{AO_2} level was investigated. It has been shown (Ernsting, 1963) that following decompression from 8,000 ft to 40,000 ft in 1.5 sec, when 100% oxygen is delivered at the beginning of the decompression, a minimum concentration of 40% oxygen must be breathed at 8,000 ft in order to present eeg changes. It can be calculated (Ernsting, McHardy and Roxburgh, 1960) that under these conditions the P_{AO_2} will fall to a minimum value of 30 mm Hg. It was decided therefore that the relationship between the area on the P_{AO_2} - time plot below a P_{AO_2} of 30 mm Hg and the variance index of the eeg (8-16 Hz) should be investigated. This relationship is presented in Fig 5 for the eeg activity recorded from the frontal pair of electrodes. There is a good linear correlation ($r = 0.85$; $p < 0.001$). A less satisfactory correlation was found between the variance index obtained from the middle channel of the eeg and this expression of the intensity of the hypoxia, whilst there was a poor correlation between the eeg activity recorded from the posterior channel and the area on the P_{AO_2} - time plot.

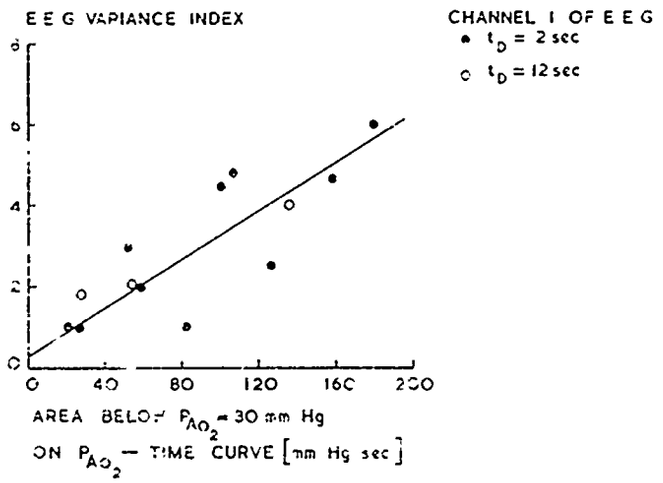

Fig 2. The relationship for individual exposures between the intensity of hypoxia induced by rapid decompression breathing air and the associated change in the frontal channel of the eeg. The intensity of the hypoxia induced by the decompression is expressed as the area below an alveolar P_{O_2} of 30 mm Hg on the plot of alveolar P_{O_2} against time whilst the change in eeg activity is expressed as the variance index for the 8 - 16 Hz band of the frontal eeg. The equation of the regression line is $y = 0.03x + 0.21$ ($r = 0.85$).

The values of the areas on the P_{AO_2} levels of 25 and 35 mm Hg were also determined and the relationships investigated between these measures of the intensity of the hypoxia and the corresponding variance indices obtained from the frontal channel. It was found that using areas below either P_{AO_2} = 25 or 35 mm Hg gave a much less satisfactory correlation than when the area below a level of P_{AO_2} = 30 mm Hg was used. Thus, under the conditions of these experiments there is a very significant correlation between the degree of hypoxia expressed as the area on a P_{AO_2} - time plot below a P_{AO_2} of 30 mm Hg and the magnitude of the consequent changes in the variance index for the 8-16 Hz band of the frontal channel of the eeg.

Previous workers (Meyer and Watty, 1961) who have studied the effects of acute hypoxia upon the eeg have placed emphasis on the appearance of the bilateral large amplitude slow wave activity which begins in the frontal regions of the head and progresses posteriorly. Whilst this type of activity was seen eventually in the present study under those conditions in which the hypoxia was most severe, ie rapid decompression with the late delivery of oxygen, the initial change of eeg activity was always the appearance of increased activity in the 8-16 Hz band.

Effect of Rate of Decompression

The effect of the rate of decompression upon the intensity of the hypoxia induced by rapid decompression from 8,000 ft to 40,000 ft can be assessed from the changes of P_{AO_2} (Fig 2), the symptoms reported by the subjects (Table 1), and the eeg changes (Table 4). The mean values of all these measures following rapid decompression show that prolongation of the time of decompression from 1.6 sec to 12.0 sec decreased the intensity of the hypoxia whether oxygen was delivered to the mask immediately the decompression commenced or after a delay of 8 sec.

There was no significant difference between the times at which the first inspiration of oxygen was taken after the beginning of the

rapid decompression in the fast and slow decompressions (Table 4). Thus the greater intensity of hypoxia which occurred in the fast decompressions is directly attributable to the more rapid fall of environmental pressure, and hence P_{AO_2} (Fig 2), which occurred with this rate of decompression.

There were two occasions in the group of slow decompressions in which the intensity of the hypoxia exceeded the mean values obtained with the corresponding nominal oxygen delivery times in the group of fast decompressions. On these occasions however the intervals between the beginning of the decompression and the first inspiration of oxygen were the longest which occurred in the series. This finding emphasizes the influence of the time interval, between the breathing equipment being capable of delivering 100% oxygen and the next inspiratory effort by the subject, upon the intensity of the hypoxia induced by a rapid decompression from 8,000 ft to 40,000 ft when air is breathed before the decompression.

The intensity of the hypoxia induced by rapid decompression in 12.0 sec from 8,000 ft to 40,000 ft when air was breathed beforehand was considerable. Thus most of the subjects reported mental confusion and some also had dimming of vision (Table 1). The hypoxia was also sufficient to produce significant increases of the variance index (8-16 Hz) of the eeg (Table 6). Furthermore, the area on a P_{AO_2} - time plot below a P_{AO_2} = 30 mm Hg was greater when oxygen was delivered late in the 12.0 sec decompression series than when oxygen was delivered immediately in the 1.6 sec decompression experiments (Table 7). It is concluded, therefore, that increasing the duration of the decompression from 1.6 sec to 12.0 sec does not prevent significant hypoxia being produced by the decompression even if oxygen is delivered within 8 sec of the beginning of the decompression.

SUMMARY

1. Three subjects were each decompressed from 8,000 ft to 40,000 ft under six different experimental conditions, viz duration of rapid

decompression of either 1.6 sec or 12.0 sec and breathing either 100% oxygen throughout the procedure or breathing air before the decompression with oxygen delivered to the mask either immediately or 8.0 sec after the beginning of the rapid decompression.

2. The intensity of the hypoxia produced by these decompressions was assessed from the subject's symptoms, the changes of the partial pressure of oxygen in the alveolar gas (P_{AO_2}) and of the eeg.

3. When air was breathed before the decompression a decompression time of 12.0 sec induced a less severe although still marked degree of hypoxia than occurred with a decompression time of 1.6 sec.

4. It was found that under the conditions of these experiments there was a linear correlation ($r = 0.85$; $p < 0.001$) between the intensity of the hypoxia as measured by the area on a P_{AO_2} - time plot below a P_{AO_2} = 30 mm Hg and the associated increase in activity (variance index) of the 8-16 Hz band of the eeg recorded from the frontal and middle regions of the head.

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